EXHIBIT 19

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Qualifications: I am a physician with board certification in both Occupational and Environmental Medicine and Internal Medicine. I received my medical degree from the State University of New York at Stony Brook, and have held faculty positions at the Schools of Medicine at Albert Einstein, Yale and George Washington Universities.

I have extensive experience in the diagnosis, epidemiology and treatment of asbestos-related diseases. I have been in occupational medicine practice for over 25 years, and a substantial part of my practice has always been devoted to examination of workers exposed to asbestos.

In addition, I have many years of experience in medical surveillance programs for asbestos. Since 1987 I have been the medical advisor to the Sheet Metal Occupational Health Institute Trust (SMOHIT), a joint labor-management organization within the sheet metal industry established to provide medical examinations for sheet metal workers exposed to asbestos and other respiratory hazards. To date, SMOHIT has provided medical examinations to over 20,000 sheet metal workers, and is now the largest epidemiological database of asbestos-exposed workers in the country. I also developed similar medical screening programs for the Laborers National Health and Safety Fund and other construction trades, in conjunction with the Occupational Health Foundation. I currently serve as medical director for a Department of Energy-funded medical screening program to provide medical examinations for former construction workers at a number of former atomic weapons production facilities. In each of these programs I have designed procedures for detection of asbestos-related disease, and designed algorithms for the examining physicians to use in interpretation of the results. I have been active in efforts to improve validity and reliability of x-ray reading to detect asbestos related disease in the United States; this work included publication of a paper on variability between readers' classification of x-rays using the International Labor Organization Guide to Classification of Pneumoconiosis, based on an analysis of results from these screening programs [(1)].

I am currently medical director at CPWR: The Center for Construction Research and Training, a research institute devoted to improving health and safety in the construction industry. Because of my expertise in medical programs for asbestos-exposed workers, I participated in a working group with representatives from labor, industry, and insurance companies to develop medical criteria for Senate Bill 1125 and later versions, a bill that proposed to establish a national trust fund for compensation of asbestos related disease in the United States. I testified before the Senate Judiciary committee on three occasions, speaking about the epidemiology and diagnosis of asbestos-related diseases, and the rationale for the medical criteria proposed for the fund

Attached as Exhibit 1 is a true and correct copy of my current curriculum vitae, which sets forth my education, training, professional affiliations, research activities and publications.

Data considered: In reaching the opinions set forth in this report, I am relying upon my background, training, and experience; the literature cited below; and generally available medical knowledge about asbestos-related diseases. I include by reference the opinions I have expressed in my expert reports and testimony in the WR Grace bankruptcy proceedings.

In addition to the literature cited I have reviewed:

- Libby Claimants' Objection To Debtors' Disclosure Statement For Joint Plan of Reorganization And Approval Motion, filed 10/17/08
- Whitehouse AC, Black CB, Heppe MS, Ruckdeschel J, Levin SM. Environmental exposure to Libby asbestos and mesotheliomas. Am J Ind Med. 2008 Nov;51(11):877-80.
- Whitehouse AC. Asbestos-related pleural disease due to tremolite associated with progressive loss of lung function: serial observations in 123 miners, family members, and residents of Libby, Montana. Am J Ind Med. 2004 Sep;46(3):219-25.
- Sullivan PA. Vermiculite, respiratory disease, and asbestos exposure in Libby, Montana: update of a cohort mortality study. Environ Health Perspect. 2007 Apr;115(4):579-85
- Expert reports of Alan Whitehouse MD dated September 25, 2006, May 8, 2007 and July 23, 2007 in this matter; and expert report of Arthur Frank MD dated September 26, 2006 in this matter, with accompanying exhibits and subsequent rebuttal reports
- Expert reports of William Longo PhD dated September 15, 2006 and July 24, 2007

Compensation: My fee schedule is attached as exhibit 2.

Prior testimony: A listing of all cases within the past four years in which I have testified as an expert at trial or at deposition is attached as exhibit 3.

THE LEGACY OF ASBESTOS

Decades of uncontrolled use of asbestos, even after its hazards were known, have resulted in an occupational disease crisis both in the United States and throughout the world of monumental scope. In 1982, William Nicholson at the Mt. Sinai School of Medicine published a detailed projection of expected cases of cancers due to asbestos exposure from 1967 to 2030 [(2)]. He estimated that in this country, from 1940 to 1979, 27.5 million workers were occupationally exposed to asbestos in shipyards, manufacturing operations, construction work and a wide range of other industries and occupations; 18.8 million of these had high levels of exposure. As a result hundreds of thousands of workers and their family members suffered from or died of asbestos-related cancers and lung disease, and more than a million more cases of malignant and non-malignant disease are expected. Because of the long lag between exposure to asbestos and the development of an asbestos related cancer or another asbestos disease, the asbestos disease epidemic reached a peak only recently in the US, and will be with us for decades to come.

Groups known to be at highest risk at the time of the Nicholson report were insulators, shipyard workers (many who worked during World War II) and workers engaged in the manufacture of asbestos products [(3-6)]. Other high- risk industries and occupations included other construction trades, railroad engine repair, utility services, stationary engineers, chemical plant and refinery maintenance, automobile maintenance and marine engine room personnel.

Many of these workers were in the group sometimes referred to as the “first wave” of asbestos exposed workers – those directly involved in the manufacture or installation of asbestos insulation or products before there were any control measures or standards in place [(7)]. Exposures for some of these workers regularly exceeded 20 – 40 fibers/cubic centimeter (f/cc), levels that are 200 – 400 times the current OSHA standard of 0.1 f/cc, with exposures of several months resulting in an increased risk of mesothelioma and lung cancer. The 1982 Nicholson analysis projected that the occupational exposures that occurred between 1940 and 1979 would result in 8,200 – 9,700 asbestos related cancer deaths annually, peaking in 2000 and then declining but remaining substantial for another 3 decades. Overall, the Nicholson study projected that nearly 500,000 workers would die from asbestos related cancers between 1967 and 2030.

It is important to point out that these projections did not include illness or death from non-malignant asbestos diseases, which have or will affect an even greater number of workers. Nor do these projections reflect the full risk of disease among populations who were exposed in the 1950s and 1960s but didn’t have sufficient latency for asbestos related diseases to be manifested at the time the Nicholson study was conducted. This includes many of the building trades and construction workers who not only installed asbestos products, but also were exposed during removal, demolition, and renovation; this group is often referred to as the “second wave” of asbestos exposed workers. Similarly, the Nicholson study did not address the risk of exposures that occurred after 1979. While, OSHA and EPA regulations reduced asbestos exposures in the 1970’s, strict regulation of asbestos did not occur until 1986. Even today, some workers, particularly those in the construction industry, may be exposed to levels of asbestos that place them at increased risk of disease if work is performed without appropriate controls.

Due to the long delay between exposure to asbestos and the onset of most asbestos related diseases (this latency can be over 40 years), many of the cases of disease today are occurring among workers who were first exposed in the 1940s, 1950s and 1960s, before asbestos was regulated and controlled.

Nicholson's work provides an excellent foundation for estimating the future cases of asbestos disease, and has been utilized in many of the models to develop future asbestos disease claims projections, including claims projections made by consultants to the Manville Trust. His estimates of mesothelioma and the of time course of the disease curve have been shown to be generally accurate when actual mesothelioma incidence and mortality is compared to his projections developed decades ago [(8)]. See also www.seer.cancer.gov.

It is important to recognize however that there is a good deal of uncertainty associated with these projections, reflected in the wide range of future disease projected by the Manville Trust and others. There are a number of factors responsible for this uncertainty. As noted above, the Nicholson study and model projected cancer mortality related to asbestos. There have been no similar studies or estimates made for the non-malignant asbestos related diseases, such as asbestosis. All of the estimates in the projections for future disease and future claims for non-malignant disease have been based upon ratios of non-malignant disease to lung cancer cases or claims, not independent estimates of non-malignant disease. Epidemiological evidence shows that hundreds of thousands of workers have developed and will develop non-malignant disease. While we know that certain groups of workers are at increased risk, and that the risk of disease will decrease as a result of reduced exposures, the extent and magnitude of non-malignant asbestos disease is not as well defined as the malignant diseases.

The most recently available Federal report from the National Institute for Occupational Safety and Health (NIOSH) shows that non-malignant lung disease from asbestos is still causing a significant number of deaths in this country [(8)]. Exhibit 4 is taken from this report, and shows an increase in the number of deaths due to asbestosis from 1968 to 2004. Although some of the increase in deaths from asbestosis is likely due to increasing recognition of asbestos as a cause of serious lung disease and therefore lung disease deaths, the number of deaths in 2004 is a significant finding in its own right.

There are several medical diseases that occur as a result of asbestos exposure. The ones of greatest concern and importance are mesothelioma, lung cancer, asbestosis, and pleural plaques and thickening. For many workers, these diseases are disabling or fatal. Below I describe the important characteristics of each medical definition in the Grace Trust Distribution Procedures (TDP) and explain why it is my opinion that the criteria for compensation for each specific medical condition are medically reasonable.

Exposure requirements:

Latency: Every category requires a minimum of 10 years since first exposure to asbestos, as described in section 5.7(a)(1) of the TDP. It is my opinion that this is medically reasonable. The time between first exposure and onset of disease is called latency; the latency for both non-malignant and malignant asbestos-related diseases is generally considered to be more than 10 years, but can be much longer. In the Selikoff studies of insulators, many cases had a latency for mesothelioma and lung cancer longer than 40 years [6;9;10]. Latency has been increasing since Selikoff's studies; in Scandinavia mean latency for a case series of mesothelioma was 45 years [11] and Miller reported in 2005 that the latency for mesothelioma from household exposure was over 40 years for the majority of cases. Relatively lower exposures have longer latency; among workers in a British naval yard the latency for mesothelioma among the heavily exposed groups was 42 years, versus 49.5 for the less heavily exposed [12]. In Miller's study the latency for some cases was over 70 years [13]; there is no upper bound on the latency of mesothelioma [14].

Exposure: In addition to the 10 year minimum latency, to meet the presumptive exposure requirements of expedited review set forth in the TDP the claimant must show:

(i) for all disease levels, Grace exposure as defined in Section 5.7(b)(3)

(ii) for asbestosis/pleural disease Level II, 6 months Grace exposure plus, for certain types of Grace exposure, five years cumulative occupational asbestos exposure; and

(iii) for asbestosis/pleural disease (disease level III), severe asbestosis (disease Level IV-A), severe disabling pleural disease (disease level IV-B), other cancer (disease level V) or lung cancer 1 (disease level VII), the claimant must show 6 months Grace exposure plus, for certain types of Grace exposure, significant occupational exposure to asbestos.

(If the claimant cannot meet these relevant presumptive exposure requirements, the claimant may seek individual review as described in the TDP).

Grace exposure: For all disease levels the claimant must demonstrate Grace exposure (as defined in Section 5.7(b)(3)):

(i) meaningful and credible exposure, which occurred prior to December 31, 1982, to

(a) any products or materials containing asbestos that were manufactured, sold, supplied, produced, specified, selected, distributed or in any way marketed by Grace (or any past or present Grace affiliate, or any of the predecessors of Grace or any of their past or present affiliates, or any other entity for whose products or operations Grace allegedly has liability or is otherwise liable) *or*

(b) asbestos-containing winchite asbestos or asbestos-containing vermiculite mined, milled or processed by Grace (or any past or present Grace affiliate, or any of the predecessors of Grace or any of their past or present affiliates, or any other

entity for whose products or operations Grace allegedly has liability or is otherwise liable) *or*

(ii) meaningful and credible exposure which occurred prior to the effective date [of the Plan] to

(a) asbestos, asbestos-containing winchite asbestos or unexpanded asbestos-containing vermiculite ore in Lincoln County, Montana *or*

(b) asbestos, asbestos-containing winchite asbestos or asbestos-containing vermiculite ore from Lincoln County, Montana during transport or use prior to the completion of a finished product at an expansion plant ("Grace Exposure").

These criteria require evidence that exposure to ore from the vermiculite mine in Libby MT or an asbestos-containing product manufactured by Grace was meaningful and credible. In my view such exposure can constitute a substantial contributing cause of the asbestos-related diseases defined in levels I-VIII. It is my opinion that these exposure criteria in the TDP are medically reasonable.

Significant occupational exposure: For disease levels III-VII the claimant must show 6 months Grace exposure plus, for certain types of Grace exposure, "significant occupational exposure" to asbestos. Significant occupational exposure means employment for a cumulative period of at least five (5) years with a minimum of 2 years prior to December 31, 1982, in an industry and an occupation in which the claimant:

(a) handled raw asbestos fibers on a regular basis;

(b) fabricated asbestos-containing products so that the claimant in the fabrication process was exposed on a regular basis to raw asbestos fibers;

(c) altered, repaired or otherwise worked with an asbestos-containing product such that the claimant was exposed on a regular basis to asbestos fibers; or

(d) was employed in an industry and occupation such that the claimant worked on a regular basis in close proximity to workers engaged in the activities described in (a), (b) and/or (c).

Overall it is my opinion that the requirement for significant occupational exposure is medically reasonable.

In 1997 a meeting of experts was convened in Helsinki to agree upon criteria both for the diagnosis of disorders of the lung and pleura associated with asbestos and attribution to asbestos exposure; the consensus document is referred to as the Helsinki criteria [(15)]. These criteria conclude that 1 year of heavy exposure (e.g., manufacturing of asbestos products, asbestos spraying, insulation work with asbestos materials, demolition of old buildings), or 5-10 years of moderate exposure (e.g., construction shipbuilding) may increase the risk of lung cancer 2 fold or more. This report concluded that the exposures described were estimated to represent a cumulative exposure of 25 fiber-years, and that clinical cases of asbestosis occur at similar cumulative exposure levels. The requirements for significant occupational exposure set forth in the TDP establish a level of exposure similar to, or higher than, those described by the Helsinki criteria for a doubling of lung cancer risk or sufficient to cause clinical asbestosis. Overall it is my opinion that these exposure criteria are medically reasonable.

Workers who handled raw asbestos fibers, manufactured asbestos products, or were asbestos insulators are required to have 5 years of occupational exposure under the TDP while the Helsinki consensus says 1 year of exposure could be sufficient to cause asbestosis or lung cancer; in my opinion the significant occupational exposure requirements may be too stringent for these groups of workers, and they should take advantage of the individual review provision. It should be noted that there are likely to be a relatively small number of workers who handled raw asbestos fibers, manufactured asbestos products, or were asbestos insulators among all the workers exposed to Grace products.

Mesothelioma (Level VIII) requires (1) Diagnosis of mesothelioma; and (2) Grace Exposure as defined in Section 5.7(b)(3)

Mesothelioma is a rare cancer of the pleura, the lining of the lung, and the peritoneum, the lining of the abdomen, that occurs in persons exposed to asbestos [16]. Mesothelioma can result from a limited exposure to asbestos, such as living with a worker exposed to asbestos at work [13;17]. All types of asbestos fibers cause mesothelioma [18-27]. There is no "safe" or threshold dose of asbestos; low dose asbestos exposures (<1 f/ml-yr) can cause mesothelioma, and epidemiology shows the risk of mesothelioma rises as asbestos dose increases. Any non-trivial asbestos exposure contributing to the cumulative dose can cause or contribute to the cause of mesothelioma [28-36].

The age of death from mesothelioma is increasing [8] In France the mean age for diagnosis was 70 between 1998 and 2002. Hodgson reports cases of mesothelioma in men over age 90. He also reported the highest death rates from mesothelioma were among men born around 1940; these men will not reach age 90 until 2030 [37].

Virtually all of mesotheliomas in this country are caused by past exposure to asbestos. Asbestos exposure causes mesothelioma not just among workers who handle asbestos and asbestos-containing products, but also among bystanders (workers who work in the vicinity of those using asbestos and asbestos-containing products), household members of asbestos-exposed workers, and communities near asbestos mines and manufacturing plants [13;31;33;38-46]. In my opinion the criteria set for mesothelioma are medically reasonable.

Lung Cancer 1 (Level VII) requires (1) Diagnosis of a primary lung cancer plus evidence of an underlying bilateral asbestos-related nonmalignant disease, (2) six months Grace exposure, (3) for claimants whose Grace exposure is not described in clause (ii) of the definition of Grace exposure, significant occupational exposure to asbestos, and (4) supporting medical documentation establishing asbestos exposure as a contributing factor in causing the lung cancer in question.

Lung Cancer 2 (Level VI) requires (1) Diagnosis of a primary lung cancer; (2) Grace exposure, and (3) supporting medical documentation establishing asbestos exposure as a contributing factor in causing the lung cancer in question. Lung Cancer 2 (Level VI) claims are claims that do not meet the more stringent medical and/or exposure requirements of Lung Cancer 1 (Level VII) claims. All claims in this disease level shall be individually evaluated.

Evidence of "Bilateral Asbestos-Related Nonmalignant Disease," for purposes of meeting the criteria for establishing Disease Levels I, II, III, V, and VII, means either

(i) a chest X-ray read by a qualified B reader of 1/0 or higher on the ILO scale or

(ii) (x) a chest X-ray read by a qualified B reader or other Qualified Physician, (y) a CT scan read by a Qualified Physician, or (z) pathology, in each case showing either bilateral interstitial fibrosis, bilateral pleural plaques, bilateral pleural thickening, or bilateral pleural calcification. (see TDP for additional details)

All major types of lung cancer are caused by asbestos. Numerous studies and expert reviews show that there is a dose-response relationship between exposure to asbestos and the risk of lung cancer, with increasing exposure leading to increasing risk of disease [6;18;47-55]. Workers with asbestosis have a higher risk than other exposed workers, but the asbestosis may simply be a surrogate measure of exposure, for significant asbestos exposure is required to cause asbestosis. Asbestosis is not a necessary intermediary for development of asbestos related lung cancer [49;53;56].

It is medically reasonable to require the presence of an underlying bilateral asbestos-related non-malignant disease to meet the presumption of the TDP, but it is well recognized that a lung cancer can be attributed to asbestos in the absence of such disease. Workers with pleural plaque do not appear to be at higher risk for lung cancer than their co-workers with similar exposure who did not develop plaque. Pleural plaque is a convenient marker of prior exposure to asbestos, and so has frequently been used as a surrogate for significant occupational exposure in bankruptcy settlement agreements. In my opinion is it important that individual evaluation be available for lung cancers that occur in the absence of an underlying bilateral asbestos-related non-malignant disease. As describe above, the Helsinki Criteria [15] established an exposure level of 25 fiber-years, or the equivalent exposure using an occupational history, as a level of exposure that significantly increases the risk of lung cancer; underlying asbestos-related non-malignant disease is not required. Several European countries have established this or a similar level of exposure as the criterion to be used for compensation of a lung cancer in an asbestos exposed worker.

In my opinion the definition of a bilateral asbestos-related nonmalignant disease is medically reasonable. The TDP relies upon the reading by a B reader or other qualified physician. A B reader is someone who is certified by the National Institute for Occupational Safety and Health (NIOSH) as competent in the use of the International Labor Organization (ILO) classification. The ILO provides a system of grading chest x-rays for dust diseases of the lung (pneumoconiosis) that is accepted around the world. The most recent version is the 2000 Classification of the Radiographic Appearance of Pneumoconioses [57]. It provides a standard notation, so that if one reader calls a film a "1/1" another reader will know to what the first reader is referring. The TDP additionally allows use of a CT scan or pathology for diagnosis.

Other Cancer (Level V) requires (1) Diagnosis of a primary colo-rectal, laryngeal, esophageal, pharyngeal, or stomach cancer, plus evidence of an underlying bilateral asbestos-related nonmalignant disease, (2) six months Grace Exposure, (3) for claimants whose Grace Exposure is not described in clause (ii) of the definition of Grace exposure,

significant occupational exposure to asbestos, and (4) supporting medical documentation establishing asbestos exposure as a contributing factor in causing the other cancer in question.

In 2006 the Institute of Medicine was charted with reviewing the evidence on the relationship between asbestos exposure and cancers other than cancer of the lung [(58)]. Cancers previously identified as caused by asbestos included pharyngeal, laryngeal, esophageal, stomach, or colorectal cancer. The committee report summarizes the review, which included a broad array of evidence from observational and experimental research. Its review emphasized epidemiologic studies of cancer rates in cohorts of asbestos-exposed workers and of risk factors in sets of individuals with cancer at the selected sites in comparison to the general population. The observational evidence was systematically identified and evaluated for its consistency and strength of association. The committee also considered the biologic plausibility of causal associations of asbestos with cancers at the specified sites, recognizing that asbestos is an established cause of mesothelioma and lung cancer. The full committee reviewed the final integration of the evidence to assure uniformity of application of the causal criteria across the sites.

Of the five sites considered, the committee found the evidence to be *sufficient* to infer a causal relationship for laryngeal cancer; to be *suggestive* for pharyngeal, stomach, and colorectal cancers; and to be *inadequate* for esophageal cancer. It is my opinion that it is medically reasonable to include all these cancers in the TDP when the criteria require significant occupational exposure to asbestos and supporting medical documentation establishing asbestos exposure as a contributing factor in causing the cancer in question. Although the IOM concluded that the evidence was inadequate for esophageal cancer, the available data is limited. There was strong evidence of increased risk of esophageal cancer with any asbestos exposure among UK asbestos-factory workers [(59)] and North American insulation workers [(4)]; in my opinion these data make inclusion of esophageal cancer medically reasonable when the TDP requirements are met.

The evidence for causality for laryngeal cancer reviewed by the IOM committee included a substantial number of both worker cohorts and general-population case-control studies, an indication of greater risk among more highly exposed persons, and the finding of an association with exposure in studies that addressed potential confounding by tobacco-smoking and alcohol consumption. In considering biologic plausibility, the committee noted that the epithelium of the larynx is similar to the respiratory epithelium lining the conducting airways of the lung. Inhaled fibers pass through the larynx and may deposit there; although fiber deposition and persistence in the larynx have not been studied extensively, there are reports of fibers and asbestos bodies being recovered from laryngeal tissues.

The committee concluded that for pharyngeal, stomach, and colorectal cancers, the case-control information was less abundant than for laryngeal cancer. For stomach and colorectal cancers, there actually were a few more informative cohorts, but fewer than half as many cohorts provided data on pharyngeal cancer as had on laryngeal cancer. The occupational-cohort studies for colon cancer suggested fairly consistently, although not uniformly, that the risk of colorectal

cancer was higher in exposed people than in the general population but there was only limited indication of exposure-response relationships.

The evidence was most sparse for cancer of the esophagus. There was a suggestion of an exposure-response relationship from the available cohort studies. Only three case-control studies were identified, and investigation of potential confounding was limited.

I have reviewed the same data reviewed by the IOM and reviewed the IOM report; it is my opinion that the criteria set by the TDP for compensation of these other cancers are medically reasonable. There is at least a potential causal relationship between asbestos exposure and these cancers, as discussed by the IOM. The TDP then requires evidence of an underlying bilateral asbestos-related nonmalignant disease, occupational exposure to asbestos as specifically defined in the TDP, and supporting medical documentation establishing asbestos exposure as a contributing factor in causing the other cancer in question. These criteria assure there has been asbestos exposure in the same range as the exposures seen in the cohort studies used to support the causal relationship. This is a reasonable approach to the uncertainty surrounding causality in any specific case.

Severe Asbestosis (Level IV-A) requires (1) Diagnosis of asbestosis with ILO of 2/1 or greater, or asbestosis determined by pathological evidence of asbestos, plus (a) TLC less than 65%, or (b) FVC less than 65% and FEV1/FVC ratio greater than 65%, (2) six months Grace Exposure, (3) for claimants whose Grace exposure is not described in clause (ii) of the definition of Grace exposure, significant occupational exposure to asbestos, and (4) supporting medical documentation establishing asbestos exposure as a contributing factor in causing the pulmonary disease in question.

The criteria used for this category of disease have been included in prior TDPs from other bankruptcies. Someone who meets these criteria has asbestosis with a high degree of certainty, since the requirement for an ILO classification of 2/1 (or asbestosis on pathology) ensures that significant fibrosis is present. The pulmonary function test criteria establish that impairment exists and that the impairment is predominately restrictive. As noted under Levels III, II, and I, individuals with asbestosis who do not meet these criteria will be included in other levels. It is my opinion that this approach is medically reasonable.

Asbestosis occurs when asbestos exposure causes scar formation in the substance of the lung itself. These scars can interfere with lung function, for they block the transport of oxygen from the air in the lungs into the blood vessels that travel through the lungs. Oxygen can only cross the membranes of the lung if they are thin; asbestosis causes them to thicken. Asbestosis also makes the lungs stiffer, which results in a decrease in lung volume and an increase in the energy needed for chest expansion. As a general rule the greater the exposure to asbestos the more likely the disease is to be present and the more severe the scarring; there is a dose-response relationship between exposure and disease. However, some people seem to form scars more readily than others, and so we see a range in the severity of disease after similar levels of exposure to asbestos.

The International Labor Organization provides a system of grading chest x-rays for dust diseases of the lung (pneumoconiosis) that is accepted around the world. The most recent version is the 2003 Classification of the Radiographic Appearance of Pneumoconioses [(57)]. It provides a standard notation, so that if one reader calls a film a "1/1" another reader will know to what the first reader is referring.

The classification uses a 12-point scale to define the degree, or severity, of increased lung markings. This scale runs from 0/- to 3/+; a "0" film is normal and a "3" film has the most severe scarring. Each reading on the scale is characterized by a number between 0 and 3, and a second number, separated from the first by "/". The first number, preceding the "/", is the final score assigned to that film by the reader. The second number, following the "/", is a qualifier. The numbers 0, 1, 2, and 3 are the main categories. An x-ray read as a category 1 film might be described as 1/0, 1/1, or 1/ 2. When the reader uses the descriptor "1/1", he is rating the film as a 1, and only considered it as a 1 film. If he uses "1/0", he is saying he rated the film as a "1", but considered calling it a "0" film before deciding it was category 1. Finally, when the reader uses "1/2", he is saying he is rating the film as a "1", but did consider calling it a "2" film. Any category "1" film is abnormal; therefore a 1/0 film in an asbestos-exposed worker is consistent with asbestosis.

Even though the ILO system was designed to standardize reading x-rays for asbestosis and other dust diseases of the lung, studies using the classification in asbestos exposed workers have found readers often disagree about classification of the same x-rays [1;60;61]. Using the classification is somewhat of an art. The "best" readers agree 80% of the time with each other; 20% of the time they assign a different score to the same x-ray. If the scarring is extensive, a difference of one grade on the scale is not important. But if the x-ray shows less extensive scarring, a difference of one grade can be the difference between making diagnosis of asbestosis or deciding asbestosis is not present. The TDP requires an ILO classification of 2/1; with this severity of scarring variability between readers is unlikely to ever result in classification of that radiograph as normal.

Brief Overview of Lung Function

The lung's primary function is to transfer oxygen from the air into the blood stream, and transfer carbon dioxide from the blood stream into the air. To accomplish this, the lung must deliver oxygen to air sacs deep in the lung (alveoli). The alveoli are located into close proximity to small blood vessels (capillaries) so that the gases can cross from lung to blood and vice versa by diffusion across a very thin membrane. Any process that thickens the membrane between the alveolus and the capillary will reduce oxygen transfer into the blood stream.

The chest around the lungs acts like a bellows. When the chest expands, the lungs are stretched; pressure inside the chest drops relative to the atmosphere outside the chest, and air is pulled in through the nose and mouth into the lungs. When the chest relaxes the lung springs back to its resting shape, expelling air out of the lungs. Any disease process in the lung that makes the lung stiffer will decrease chest expansion and so limit how much air can be inhaled with each breath, as well as increase the energy needed to breathe. Scarring of the lining of the lung in any way that interferes with chest wall motion will have the same effect.

The lung has mechanisms to defend against foreign substances, from bacteria to asbestos. Those defense mechanisms are an integral part of the disease process after asbestos exposure. Inhaled air is delivered to the alveoli along a duct system of bronchi, and the bronchial tubes are lined with mucus to trap particles before those particles penetrate into the lung. If foreign substances do reach the alveoli, scavenger cells called macrophages attach them. Macrophages engulf asbestos fibers and try to destroy them. In many cases, the fiber survives, the macrophage dies, and oxygen radicals and inflammatory substances are released into the lung. The end result is scar formation in the lung from the release of substances that promote activity of fibroblasts, the cells that lay down scar after injury.

The lung also gets rid of foreign substances through the lymphatic drainage system. Asbestos fibers are carried through the lymph system to the pleural space, and can become trapped there. Once located in the pleura space, these fibers can induce scar formation.

Pulmonary function testing

Spirometry measures lung volume and air flow with equipment that is readily available in many physicians' offices. Spirometry is reliable and reproducible when performed according to the

specifications set by the American Thoracic Society and the European Respiratory Society (ATS/ERS) [62]. The primary measures produced by spirometry are the forced vital capacity (FVC), the forced expiratory volume in one second (FEV1) and the ratio of the two (FEV1/FVC). FVC is a measure of lung volume. The FEV1/FVC ratio measures how quickly that lung volume is expelled from the lung, and so measures airflow. A reduction in FVC with a normal FEV1/FVC ratio is due to loss of lung volume, while a reduction in FEV1 with a reduced FEV1/FVC is likely due to air flow obstruction.

Total lung capacity (TLC) is a more extensive test than spirometry; it also measures lung function. Determination of lung volumes can be done by the gas dilution method or by body plethysmography; both are standard measures and also are reliable and reproducible [63]. The advantage of measuring lung volume with the TLC is this method is less dependent on the effort of the patient, and TLC measures different compartments of lung volume. The disadvantage to using TLC as part of the testing required for a compensation trust is that determination of TLC is considerably more expensive, and less available, than spirometry.

Single breath diffusing capacity (DLCO) is the test most commonly used to measure the capacity of the lung to exchange oxygen and other gases across the alveolar-capillary membrane. While spirometry and total lung capacity measure lung volume and air flow, DLCO measures a different component of lung function. DLCO is determined by many structural and functional properties, including lung volume, the thickness and area of the alveolar capillary membrane, the volume of blood in capillaries supplying ventilated alveoli, absolute levels and distribution of both ventilation and perfusion, the concentration and binding properties of hemoglobin in the alveolar capillaries, and the gas tensions in blood entering the alveolar capillaries [64]. The ATS/ERS also sets standards for diffusion capacity, which ensure uniformity and reproducibility among laboratories [64].

DLCO is not included in the TDP medical criteria for severe asbestosis. It is the case that DLCO is a routine part of the clinical evaluation of asbestosis and other interstitial lung diseases, and DLCO is recommended by the American Thoracic Society as part of the evaluation of asbestosis [65]. DLCO is also decreased in emphysema, and therefore a reduction in DLCO is less specific for interstitial lung disease than is a reduction in lung volumes [65-67]. A reduced DLCO with a normal TLC and normal FVC could be due to emphysema or some other interstitial lung disease and not asbestosis.

DLCO has higher test-to-test variation than either TLC or spirometry. The ATS/ERS document on standardization of diffusion capacity reported that in a large university-based laboratory study the coefficient of variation in repeated measurements of spirometry was 3.1% in normal subjects and 4-4.4% in subjects with abnormal spirometry. In contrast, the inter-session variation for DLCO was up to 9% in normal individuals over a 1 year period [64]. The ATS/ERS requires for there should be at least two acceptable DLCO tests that meet the repeatability requirement of either being within 3 mL CO or within 10% of the highest value. In contrast, the acceptable variability for spirometry is less than 150 ml, which in an average man is less than 3% of the highest value. Variation from test to test in a single session is primarily due to technique rather than physiology [64]. With a 10% variation acceptable in a single session, and a 9% variation possible over a year's period, one might find as much as a 19% variation from one DLCO

determination to another on different days for a single individual, even with adherence to the ATS standards.

Because the DLCO test has high variability reference values for DLCO vary much more than reference values for other lung function testing. The ATS/ERS document on interpretative strategies for lung function tests states: "Selecting reference values for DLCO is more problematic than selecting reference values for spirometry because inter-laboratory differences are much larger for DLCO. Some of these differences can be attributed to the method of calculating DLCO and adjusting for hemoglobin concentration, carboxyhemoglobin concentration and altitude. Laboratory directors should thoughtfully select reference values that match the numbers produced in their laboratories. Optimally, it would require individual laboratories to measure DLCO in a sample of healthy subjects and compare the results with several reference equations. At the very least, laboratory directors should be alert to frequent interpretations that do not match the clinical situation. Such mismatches may signal inappropriate reference values or problems with the DLCO measurement. Predicted values for alveolar volume (VA) inspired volume (VI), DLCO and transfer coefficient of the lung for carbon monoxide (KCO) should be derived from the same source…a statement should be included describing which parameters might have been used to adjust the predicted values (e.g. VA, hemoglobin and carboxyhemoglobin concentrations, and altitude)." [68]. Because of the variability in the test within and between testing days, and because reference values also vary significantly, DLCO has not been considered a reliable enough test to use as a presumption for compensation in the TDP.

FEV1/FVC ratio: When the FVC is used to measure the presence and extent of restrictive impairment, the TDP requires that the FEV1/FVC ratio must be greater than 65%. Setting the lower limit of the FEV1/FVC ratio at 65% will exclude claimants who have an obstructive defect from asthma, asbestosis or smoking. If a claimant has several co-existing medical conditions such as asbestosis and COPD from smoking, an individual evaluation would be needed to determine if the asbestosis was a substantial contributing factor to the impairment.

Severe Disabling Pleural Disease (Level IV-B) requires (1) Diagnosis of diffuse pleural thickening of at least extent "2" and at least width "a" as one component of a bilateral non-malignant asbestos related disease based on definitions as set forth in the 2000 revision of the ILO classification, plus (a) TLC less than 65%, or (b) FVC less than 65% and FEV1/FVC ratio greater than 65%, (2) six months Grace exposure, (3) for claimants whose Grace exposure is not described in clause (ii) of the definition of Grace exposure, significant occupational exposure to asbestos and (4) supporting medical documentation establishing asbestos exposure as a contributing factor in causing the pulmonary disease in question.

The pleura is a thin lining that surrounds the lung. There are two pleural layers, one on the chest wall and one on the lung, with a negative pressure relative to the atmosphere and a small amount of fluid between. The pleura allow the lung to expand easily inside the chest wall.

As noted above, asbestos fibers that are breathed into the lung are transported to the outside of the lung into the pleural space, and cause a scar to form in the pleural lining. When these scars reach a certain size they are visible on chest x ray. A majority of persons with heavy exposure to asbestos develop some kind of pleural scarring. Pleural scars are described as pleural plaques, pleural thickening, diffuse pleural thickening, pleural fibrosis, and pleural asbestosis. There is no universal agreement on the meaning of each of these terms. The 2000 revision to the ILO classification clarifies the definition of one type of scarring, diffuse pleural thickening as present "only in the presence of and in continuity with an obliterated costophrenic angle."

It is generally accepted that pleural inflammation from asbestos results in scarring on either the parietal (chest wall) pleural surface, or on the visceral (lung) pleural surface. The American Thoracic Society publication *Diagnosis and Initial Management of Nonmalignant Diseases Related to Asbestos* [(65)] describes the difference between these two types of pleural scarring:

> "***Plaques: circumscribed pleural thickening.*** Pleural plaques are indicators of exposure to asbestos. They are clearly the most common manifestation of the inhalation, retention, and biologic effect of asbestos..). Pleural plaques are bilateral, but not symmetric, lesions of the **parietal pleura**… The conventional chest film is a sensitive and appropriate imaging method for plaques, although it may identify abnormalities that resemble plaques but are not. In the PA radiograph, they are best seen in profile on the midlateral chest walls and on the diaphragm or face on, and show serrated borders… Slow progression of plaques is typical. Approximately 85% of heavily exposed workers showed pleural thickening (predominantly plaques) on plain film more than 40 years from first exposure, as did up to 17% of environmentally exposed populations… Although pleural plaques have long been considered inconsequential markers of asbestos exposure, studies of large cohorts have shown a significant reduction in lung function attributable to the plaques, averaging about 5% of FVC, even when interstitial fibrosis (asbestosis) is absent radiographically. Even so, most people with pleural plaques alone have well preserved lung function." ….
>
> ***"Diffuse pleural thickening.*** Diffuse thickening of the **visceral pleura** is not sharply demarcated and is often associated with fibrous strands ("crow's feet") extending into the

> parenchyma. In large surveys of asbestos-exposed workers, diffuse pleural thickening has ranged from 9 to 22% of those with pleural disease… **This condition affects the visceral pleural surface** and is quite different in appearance from the parietal pleural plaque. Diffuse pleural thickening may have a significantly greater impact on pulmonary function than circumscribed plaques…Decrements associated with diffuse pleural thickening reflect pulmonary restriction as a result of adhesions of the parietal with the visceral pleura. Restrictive impairment is characteristic, with relative preservation of diffusing capacity."

As noted by the ATS document, it is generally accepted that visceral pleural thickening can cause entrapment of the lung and can lead to significant reduction in pulmonary function testing. The current edition of the ILO classification requires that blunting of the costophrenic angle be present in order to describe pleural scarring as diffuse pleural thickening; for many decades experts have suggested that blunting be required since it is particularly in cases with obliteration of the costophrenic angle that restrictive lung function occurs [69-71]. Parietal, or circumscribed, plaques are also associated with a reduction in lung volumes, although to a lesser extent than that seen with visceral thickening and blunting the costophrenic angle [72-74] . Generally the effect of circumscribed plaques on lung volume reported in the literature is a decrement in the range of 1.5-5% [75]. The loss of lung volume seen with parietal plaques could be due to parenchymal asbestosis not visible on chest radiograph as well, although several of the studies cited have found that pleural scarring predicts reduced lung function while controlling for parenchymal disease in the analysis.

The criteria for the TDP require width "a" and extent 2 for diffuse pleural thickening. Width "a" is the minimum width for reporting diffuse pleural thickening using the ILO Classification. The medical criteria must be based around the ILO Classification, since this classification has been used as a standard method for description of asbestos-related disease on chest radiograph for over 50 years. The definitions of *diffuse pleural thickening, extent* and *width* must come from the 2000 ILO Classification for Pneumoconiosis. The 2000 ILO classification restricts diffuse pleural thickening to cases where there is associated blunting of the costophrenic angle; this is a change from the prior versions of the ILO classification [57].

The dimensional criteria, and the use of the ILO classification, are included to assure that diffuse thickening is present, and not as a measure of severity. Severity is defined by pulmonary function testing.

The 2000 version of the ILO Classification requires the presence of blunting of the costophrenic angle in order to classify pleural scarring as diffuse pleural thickening. As noted above, for decades experts have included blunting of the costophrenic angle as part of the criteria for diagnosis of diffuse pleural thickening [76;77]. Blunting of the costophrenic angle was the radiographic finding predictive of restrictive lung disease in a study of over 1500 asbestos workers [77]. Ameille concluded that obliteration of costophrenic angle was more reliable than any other criteria to characterize DPT [78]. The most recent revision of the ILO classification is consistent with this medical literature.

DLCO is not included in the medical criteria for severe disabling pleural disease. It is the case that DLCO is a routine part of the clinical evaluation of asbestosis and other interstitial lung diseases, and is often reduced when any interstitial lung disease is present. DLCO is also decreased in emphysema. In addition, as noted in the ATS document *Diagnosis and Initial Management of Nonmalignant Diseases Related to Asbestos* (65), restrictive impairment (reduction in lung volume) with relative preservation of diffusing capacity is characteristic of diffuse pleural thickening. A reduced DLCO in the setting of asbestos-related pleural disease would be more likely to be due to emphysema, asbestosis, or some other interstitial lung disease that was not visible on the chest radiograph than to asbestos-related pleural disease (67).

When the FVC is used to measure the presence and extent of restrictive impairment, the TDP requires that FEV1/FVC ratio must be greater than 65%. Setting the lower limit of the FEV1/FVC ratio at 65% will exclude claimants who have an obstructive defect from asthma, asbestosis or smoking. The criteria for severe disabling pleural disease are designed to compensate those who have a severe impact on lung function attributable to pleural disease, and not to compensate those who have pleural disease in combination with functional impairment caused by another medical condition. If a claimant has several co-existing medical conditions such as asbestos-related pleural disease and COPD from smoking, an individual evaluation would be needed to determine if the asbestos-related pleural disease was a substantial contributing factor to the impairment.

Asbestosis/Pleural Disease (Level III) requires (1) diagnosis of bilateral asbestos-related nonmalignant disease, plus (a) TLC less than 80%, or (b) FVC less than 80% and FEV1/FVC ratio greater than or equal to 65%, and (2) six months grace exposure, (3) for claimants whose Grace exposure is not described in clause (ii) of the definition of Grace exposure, significant occupational exposure to asbestos, and (4) supporting medical documentation establishing asbestos exposure as a contributing factor in causing the pulmonary disease in question.

This category will compensate individuals who have a non-malignant asbestos related disease on chest radiograph or CT scan in conjunction with restrictive lung disease. Individuals with asbestosis who do not meet the stricter radiographic criteria set under IV-A and IV-B fall into this category. In my opinion the criteria for this disease level are medically reasonable; people who meet these medical and exposure criteria in all likelihood have an asbestos-related non-malignant disease caused in whole or in part by exposure to Grace asbestos, even though they are not severely impaired by the disease.

Asbestosis/Pleural Disease (Level II) requires (1) diagnosis of a bilateral asbestos-related nonmalignant disease, and (2) six months Grace exposure, and (3) for claimants whose Grace exposure is not described in clause (ii) of the definition of Grace exposure, five years cumulative occupational exposure to asbestos.

This category will compensate individuals who have a non-malignant asbestos related disease on chest radiograph or CT scan, and not have characteristic restrictive lung disease. Individuals

with asbestosis who do not meet the stricter criteria set under IV-A, IV-B and III fall into this category. In my opinion the criteria for this disease level are medically reasonable; people who meet these medical and exposure criteria in all likelihood have an asbestos-related non-malignant disease caused in whole or in part by exposure to Grace asbestos, even though they are not impaired by the disease. As described in the ATS document *Diagnosis and Initial Management of Nonmalignant Diseases Related to Asbestos* (65) there is no requirement for lung function impairment to make a diagnosis of an asbestos-related non-malignant disease.

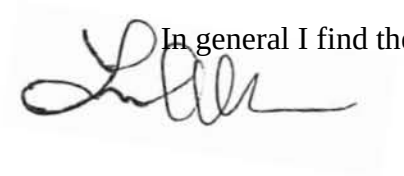
Other Asbestos Disease (Level I) requires (1) diagnosis of a bilateral asbestos-related nonmalignant disease or an asbestos-related malignancy other than mesothelioma, and (2) Grace exposure.

This category will compensate individuals who have a non-malignant asbestos related disease on chest radiograph or CT scan, and either do not have characteristic restrictive lung disease or do not have enough exposure to asbestos to be included under categories II or III. In my opinion it is medically reasonable that this category be included, for there will be individuals with asbestosis or asbestos-related pleural disease who cannot meet the criteria set for Levels IV-A, IV-B and III.

Exposure to asbestos-containing vermiculite ore from Lincoln County, Montana can and does occur outside of Libby MT, as can happen during transport of the ore or its use prior to the completion of a finished product at an expansion plant. Disease among persons exposed to the asbestos in this ore has arisen and is likely to arise in the future.

ATSDR Summarized information about exposure to asbestos-containing vermiculite from Libby, Montana, at 28 processing sites in the United States in a report published in October 2008 (79). ATSDR identified these three groups of people who experienced significant exposure to asbestos (specifically Libby amphiboles) associated with facilities that exfoliated vermiculite: former employees, household contacts of former employees, and some community members, particularly children, who had frequent, direct contact with VC or waste rock from these facilities. ATSDR concluded that these groups were exposed to asbestos in the past, when the facilities actively were exfoliating VC from Libby, and that they likely have increased risk for developing both carcinogenic and non-carcinogenic asbestos-related diseases. Rohs and colleagues found that over 30% of workers at one such vermiculite expansion plant had non-malignant asbestos-related disease on chest x-ray (80); disease from this occupational exposure had previously been described by Lockey (81).

In general I find the medical and exposure criteria of the TDP to be medically reasonable.



12/23/08

Signature Date

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